Heritability of Self-Reported Health

James C. Romeis, Jeffrey F. Scherrer, Hong Xian, Seth A Eisen, Kathleen Bucholz, Andrew C. Heath, Jack Goldberg, Michael J. Lyons, William G. Henderson, and William R. True

Objective. To explore the contribution of genes and environmental factors to variation in a common measure (i.e., a five-point—excellent, very good, good, fair, and poor—Likert scale) of self-reported health.

Data Sources. Data were analyzed from 4,638 male-male twin pair members of the Vietnam Era Twin (VET) Registry who responded to a 1987 health survey.

Study Design. Varying models for the relationship between genetic and environmental influences on self-reported health were tested in an attempt to explain the relative contributions of additive genetic, shared and nonshared environmental effects, and health conditions reported since 1975 to perceived health status.

Data Collection. A mail and telephone survey of health was administered in 1987 to VET Registry twins.

Principal Findings. Variance component estimates under the best-fitting model included a 39.6 percent genetic contribution to self-reported health. In a model which included the effect of health condition, genes accounted for 32.5 percent and health condition accounted for 15.0 percent of the variance in self-reported health. The magnitude of the genetic contribution to perceived health status was not significantly different in a model with or without health condition.

Conclusions. These data suggest over one-third of the variability of self-reported health can be attributed to genes. Since perceived health status is a major predictor of morbidity, mortality, and health services utilization, future analyses should consider the role of heritable influences on traditional health services variables.

Key Words. Genetic models, self-reported health, twins, veterans

INTRODUCTION

Behavior genetics is a relatively new scientific discipline that focuses on the etiology of individuality, or differences among individuals within populations. Heritability (Loehlin 1992) refers to the contribution of genes to individual differences in a particular trait in some particular population. Broadly speaking, the proportion of the variation in the trait, or phenotype, is accounted for by genes. Heritability studies estimate the proportion of variation attributed

to either genetic or nongenetic factors. Heritability is defined as a ratio of variances: the ratio of the genetic variance to the total phenotypic variance in the population and ranges from 0 to 1 (Khoury, Beaty, Cohen 1980). The upper limit is 1.0, or 100 percent, when all phenotypic variation is attributed to genes and 0 percent when no phenotypic variation is attributed to genes.

The synthesis of traditional approaches to health services research with behavior genetics has produced promising results as evidenced from our earlier work (True, Romeis, Heath, et al. 1997), which estimated the genetic and environmental influences on help seeking and health services use. We found that genes accounted for 24 to 52 percent of the variance in condition status and from 42 to 56 percent of the variance in treatment seeking. We now seek to apply genetic modeling techniques to estimate the genetic and/or environmental influences on a standard, global measure of self-reported health.

Self-reported health is an active process similar to other cognitive and emotional strategies used in assessing the self (Mechanic 1994). A review of the literature on either single-item or multi-item composite, self-reported health

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Address correspondence to James C. Romeis, Ph.D., Professor of Health Services Research, School of Public Health, St. Louis University, 3663 Lindell Blvd., St. Louis, MO 63108. James C. Romeis is also from Research Services, St. Louis VAMC. Jeffrey F. Scherrer, M.A. is from the Research Service, St. Louis VAMC; and Health Services Research, School of Public Health, St. Louis University. Hong Xian, Ph.D. is from the Research Service, St. Louis VAMC; and Department of Internal Medicine, Division of General Medical Sciences, Washington University School of Medicine, St. Louis, MO. Seth A. Eisen, M.D., M.Sc. is from the Research Service, St. Louis VAMC; the Medical Service, St. Louis VAMC; and the Department of Internal Medicine, Division of General Medical Sciences, Washington University School of Medicine, St. Louis, MO. Kathleen Bucholz, Ph.D. is from the Department of Psychiatry, Washington University School of Medicine, St. Louis, MO. Andrew C. Heath, D.Phil. is from the Department of Psychiatry, Washington University School of Medicine, St. Louis, MO. Jack Goldberg, Ph.D. is from the Department of Veterans Affairs, Health Services Research and Development, Vietnam Era Twin Registry, Hines, IL; and the Epidemiology Program, School of Public Health, University of Illinois, Chicago. Michael J. Lyons, Ph.D. is from the Department of Psychology, Boston University; the Harvard Medical School, Department of Psychiatry at the Brockton/West Roxbury VAMC, Brockton, MA; and the Harvard Institute of Psychiatric Epidemiology and Genetics. William G. Henderson, Ph.D. is from the Department of Veterans Affairs, Health Services Research and Development, Vietnam Era Twin Registry, Hines, IL. William R. True, Ph.D., M.P.H. is from the Research Service, St. Louis VAMC; and Health Services Research, School of Public Health, St. Louis University. This article, submitted to *Health Services Research* on April 21, 1999, was revised and accepted for publication on November 8, 1999.

measures reveals well-defined conceptual and methodological studies from the behavioral, social, and medical sciences, and one recent set of reports from the Karolinska Institutet (Harris, Pedersen, McClearn, et al. 1992; Svardh, Isacson, and Pedersen 1998; Lichtenstein et al. 1992). These studies partitioned a four-item composite, self-reported health measure, as part of their genetics and aging studies, into its genetic and environmental components.

In general, self-reported health is positively correlated with clinical assessments; and, in some instances, subjective assessments are superior relative to mortality and other outcomes controlling for demographic, social-psychological, and clinical variables (Bergner, Bobbitt, Kressel, et al. 1976; Bergner and Rothman 1987; Ferraro 1980; Mossey and Shapiro 1982; Miilunpalo, Vuori, Oja, et al. 1997; Idler and Benajamani 1997). Income, education, race, marital status, pain perception, psychiatric illness, and personality factors also strongly influence self-reported health (Nickens 1995; Barsky, Cleary, and Klerman 1992; Hidding, de Witte, and van der Linden 1994). In addition, there is a voluminous literature on measurement; see Andresen, Bowley, Rothenberg, et al. (1998); Bergner, Bobbitt, Kressel, et al. (1976); Bergner and Rothman (1987); Greenfield and Nelson (1992); Ware (1992); Anonymous (1989); McHorney, Kosinski, and Ware (1994); McHorney et al. (1994); McHorney, Ware, and Raczek (1993); and Ware and Sherbourne (1992), to mention but a few. More recently, Malmstrom, Sundquist, and Johansson (1999) report that socioeconomic neighborhood is related to self-reported health; and Kawachi, Kennedy, and Glass (1999) cast self-reported health into a social capital framework.

Many factors that influence self-reported health are also familial. Familial traits result from influences from genes and family environmental experiences shared by siblings in childhood typically. Familial factors have been shown to contribute to socioeconomic status variables (Heath et al. 1987; Taubman 1978), pain perception (McGrath 1994), and personality traits (Loehlin 1992). Familial factors may contribute to self-reported health via shared family experiences or genes that influence pain perception, personality, and socioeconomic variables that affect health status.

The Karolinska Institutet studies use a standardized, four-item measure of subjective, self-reported health similar to the General Health domain of the SF 36 (Ware and Sherbourne 1992) with the Swedish Adoption/Twin Study of Aging (SATSA). The Institutet's model-fitting analysis reveals no genetic influences on younger cohorts [< 60]; 75 to 77 percent of the variance attributed to unique or nonshared environment factors and error; and 23 to 25 percent of the variance attributed to correlated environment factors, or

post-rearing shared environmental influences. In the older cohorts, genetic factors account for 26 to 29 percent of the variance of subjective health with the remainder attributed to nonshared environmental factors. For both age cohorts, no shared environment effects (e.g., early childhood experiences) were identified. We believe that this report is the first heritability analysis of the single item, self-reported health measure.

METHODS

We examined the genetic and environmental contributions to the variation in response to one of the most common measures of self-reported health in the field—a single-item, five-point Likert-type scale that asks *Would you say that your health in general is excellent, very good, good, fair, or poor?* Data were collected from monozygotic and dizygotic twin pair members of the Vietnam Era Twin (VET) Registry who responded to the VA's Survey of Health in 1987. First, univariate estimates were calculated for self-reported health to measure the magnitude of the contribution of genes and environment to this variable. Second, heritability estimates were reexamined accounting for the variance that resulted from significant self-reported health conditions experienced since 1975 or discharge. The survey did not provide a specific date and does not permit an estimate of symptom or treatment status at the time of survey.

Subjects and Data Collection

The Vietnam Era Twin (VET) Registry is a national sample that comprises twins who served in the military during the Vietnam era (May 1965–August 1975). The Registry contains 7,375 male-male monozygotic (MZ) and dizygotic (DZ) twin pairs born between 1939 and 1957. It was assembled from computerized military records provided by the Department of Defense and several civilian databases using an algorithm that matched database entries for the same last name, same date of birth, and similar social security number. Complete descriptions of zygosity determination and characteristics of registry members have been published (Eisen, True, Goldberg, et al. 1987; Eisen, Neuman, Goldberg, et al. 1989; Henderson, Eisen, Goldberg, et al. 1990).

Registry members were surveyed in 1987, a mean of 19 years after induction into service. A 30-page Survey of Health was mailed and administered to all VET Registry twins (one percent of interviews were conducted by telephone as a follow up to nonresponse). The survey was designed to

collect data on sociodemographics, service in Vietnam, combat exposure, major health conditions such as high blood pressure since discharge, health care utilization, smoking and drinking status, mental health status, and self-reported health. The overall case-wise response rate was 74.4 percent, and both brothers of a twin pair (i.e., pairwise) responded at a rate of 64.4 percent. Reasons for nonresponse included refused (9.5 percent), death (2.7 percent), ineligible (0.4 percent), unavailable for study (either outside United States or too ill—0.5 percent), and no response after repeated calls (12.4 percent). According to Henderson, Eisen, Goldberg, et al. (1990), respondents were more likely to be white (77.4 versus 57.3 percent of nonwhites), have at least a high school diploma (83.5 percent versus 62.3 percent of nongraduates), were 20 years of age or older at time of enlistment (78.5 percent versus 72.0 percent younger than 20), and to have served in Southeast Asia (77.7 percent versus. 74.4 percent who served elsewhere). Goldberg, True, Eisen, et al. (1987) report that ascertainment bias was found for military service variables (e.g., year of discharge, branch of service, length of military service), but none related to their physical or psychosocial health.

The final sample contained 4,638 twin pairs, (2,551 MZ; 2,087 DZ). The mean age of the twins in 1987 was 38.1 (SD $\pm$ 2.8). They were generally well educated—32 percent were high school graduates, 29 percent had completed two years of college, 12 percent were college graduates, and 11 percent reported some graduate study. Most (74.3 percent) respondents were married, 12 percent divorced, 1 percent separated, and 11 percent never married. Nearly all (91 percent) were employed full time, 2 percent employed part time, and 7 percent were not employed. Eighteen percent had annual household incomes less than $20,000; 24 percent between $20,001 and $30,000; 25 percent between $30,001 and $40,000, and 34 percent reported annual incomes greater than $40,000. Ninety-three percent were classified as white, 6.5 percent black, and 0.5 percent classified themselves as "other."

Analytic Strategy

The genetic model fitting was performed to determine whether genetics, family environment, unique environmental factors, and the report of a significant health problem since discharge influence self-reported health. We also calculated the magnitude of the influence of these factors for the model that best explained the variability in self-reported health.

Twin siblings were randomly assigned to index and co-twin status. For each zygosity group, each twin's level of self-reported health was cross-classified with the co-twin's level of self-reported health and summarized in

a 5 × 5 contingency table. For the ordinal variable "self-reported health," polychoric correlations (Olsson 1979) were calculated for both MZ and DZ pairs. The correlation is a measure of similarity between the members of MZ twin pairs and the members of DZ twin pairs and is calculated assuming an underlying bivariate normal distribution. If the MZ correlation coefficient is twice the DZ correlation coefficient, then additive genetic effects likely influence self-reported health. If the MZ correlation is approximately equal to the DZ correlation, then shared environmental influences likely account for variance in self-reported health. Finally, if the correlation for both pairs is low, then nonshared or unique environment, which includes error, would be expected to account for the variance in self-reported health.

A full univariate model was fit to the observed MZ and DZ correlations by the method of maximum likelihood. The full model consists of additive genetic (notated as A most likely multiple loci), shared environmental (notated as C), and unique environmental factors (notated as E). The latter includes measurement error. The modeling was extended to allow for the variability in health status to be partially a result of self-reported physical conditions. In this case, the full model consisted of additive genetic, shared environmental, and unique environmental factors, and the influence of significant self-reported health conditions (B) since 1975 or discharge. The examination of health conditions functions similar to a control variable.

Model fitting was utilized to resolve competing hypotheses about the genetic and environmental influences to self-reported health status and to identify the most parsimonious model consistent with the data (Neale and Cardon 1992). For example, one hypothesis is that self-reported health is entirely a result of unique environmental effects—that is, environmental influences of one twin are independent of those on the other twin (random effects), or E. Another competing hypothesis we tested is that self-reported health is influenced by both genes and unique environment, or AE. A third competing hypothesis is that self-reported health is entirely a result of experiences shared by twins plus the unique environment, or CE. A final hypothesis is that self-reported health is a result of a combination of genes, shared environment, and unique environment, or ACE. Model fitting is, therefore, used to resolve which hypothesis best explains the variance in self-reported health. Nested models were compared by likelihood-ratio Chi-square. Degrees of freedom are equal to the number of parameters deleted from the full model. Reduced models that removed the influence of one or more factors (e.g., additive genetic effects) were compared to the fit of the full model. If a reduced model provided as good a fit or a better fit than the full model, then the

simpler model was selected as best fitting. If two or more reduced models provided an equally good fit, then the most parsimonious model was selected by Akaike's Information Criterion (AIC) (Akaike 1987). The AIC is calculated by subtracting two times the degrees of freedom from χ^2. The model with the lowest AIC was identified as best fitting the data.

Detailed genetic modeling procedures are described in Neale and Cardon (1992). MX software (Neale 1994) was used for the genetic modeling and PRELIS 2 (Joreskog and Sorbom 1988) was used to compute tetrachoric correlations and asymptotic covariance matrices.

RESULTS

Table 1 is the polychoric correlation coefficient matrix for self-reported health among the MZ and DZ twins. The top portion represents the cross-tabular distributions and the polychoric correlation coefficient for the MZ twins. The bottom portion represents the same information for the DZ twins. The magnitude of the correlations suggests neither a single genetic or environmental explanation. The MZ correlation coefficient is approximately twice the magnitude for the DZ coefficient, which suggests additive genetic influences on the variability in self-reported health.

Table 2 indicates the frequency distribution of self-reported health conditions since 1975 or discharge. Approximately two-thirds of the respondents reported suffering one or more health conditions since leaving the military. The prevalence of conditions ranged from 0.3 percent for stroke to 16.3 percent for high blood pressure.

Univariate model-fitting results are shown in Tables 3a and 3b. In Table 3a, the full ACE model that allowed for additive genetic, shared environmental, and unique environmental influences to self-reported health provided an adequate fit to the data. However, a model that did not allow for shared environmental influences was selected as the best-fitting model for the variability in self-reported health. This AE model provided a more parsimonious fit to the data and did not result in a worse fit compared to the full model ($\Delta\chi^2_1 = 0$, AIC $= -0.514$).

As shown in Table 3b, the full ABCE model that allowed for the variance in self-reported health to be as a result of additive genetic factors, the presence of one or more significant self-reported physical health conditions since discharge, and shared and unique environmental influences provided an adequate fit to the data ($\chi^2 = 9.845$, $p = .363$, AIC $= -8.155$). A model that

Table 1: Polychoric Correlations and Twin Pair Contingency Table for a Five-Level, Self-Reported Health Measure.

Monozygotic pairs (n = 2,551); polychoric correlation = 0.403

			Twin 2			
Twin 1	*Excellent*	*Very good*	*Good*	*Fair*	*Poor*	*Total*
Excellent	485 (19.0%)	325 (12.7%)	106 (4.2%)	17 (0.7%)	0 (0.0%)	933 (36.6%)
Very good	310 (12.2%)	478 (18.7%)	190 (7.5%)	33 (1.3%)	9 (0.4%)	1020 (40.0%)
Good	103 (4.0%)	191 (7.5%)	164 (6.4%)	30 (1.2%)	3 (0.1%)	491 (19.2%)
Fair	10 (0.4%)	30 (1.2%)	26 (1.0%)	19 (0.7%)	2 (0.1%)	87 (3.4%)
Poor	2 (0.1%)	3 (0.1%)	2 (0.1%)	5 (0.2%)	8 (0.3%)	20 (0.8%)
Total	910 (35.7%)	1027 (40.3%)	488 (19.1%)	104 (4.1%)	22 (0.9%)	

Dizygotic Pairs (n = 2,087); polychoric correlation = 0.168

			Twin 2			
Twin 1	*Excellent*	*Very good*	*Good*	*Fair*	*Poor*	*Total*
Excellent	261 (12.5%)	270 (12.9%)	126 (6.0%)	26 (1.2%)	1 (0.01%)	684 (32.8%)
Very good	256 (12.3%)	351 (16.8)	187 (9.0%)	39 (1.9%)	9 (0.4%)	842 (40.3%)
Good	105 (5.0%)	185 (8.9)	121 (5.8%)	19 (0.9%)	5 (0.2%)	435 (20.8%)
Fair	26 (1.2%)	37 (1.8%)	29 (1.4%)	16 (0.8%)	2 (0.01%)	110 (5.3%)
Poor	3 (0.1%)	8 (0.4%)	3 (0.1%)	1 (0.01%)	1 (0.01%)	16 (0.8%)
Total	651 (31.2%)	851 (40.8%)	466 (22.3%)	101 (4.8%)	18 (0.9%)	

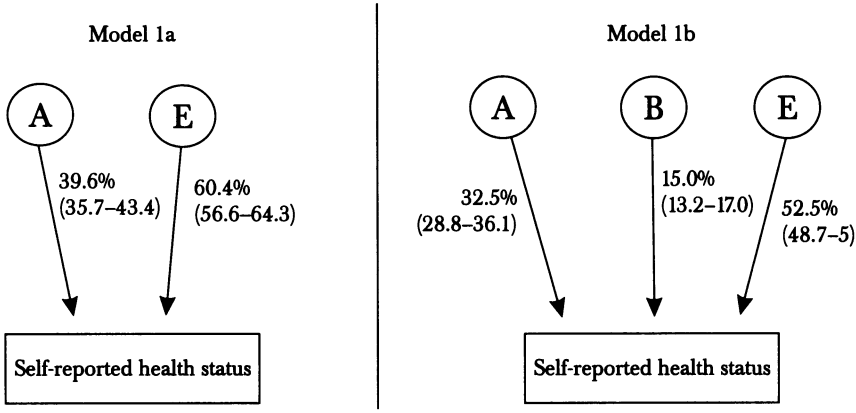
Table 2: Frequency Distribution of Self-Reported Health Conditions Reported Since 1975 or Discharge

Condition	*% Yes*
Any problem	63.0
High blood pressure	16.3
Stomach disorders	16.0
Joint disorders	15.8
Hearing problems	15.6
Skin problems	13.1
Kidney problems	10.9
Respiratory condition	10.0
Nerve disorders	5.7
Heart trouble	3.2
Liver disorders	2.4
Blood disorders	1.5
Diabetes	1.2
Cancer	1.0
Stroke	0.3
Other health problems	25.5

did not allow for shared environmental contributions to self-reported health (ABE) was chosen as best fitting since it provided a more parsimonious fit to the observed data and was not worse fitting than the full model ($\Delta\chi_1^2 = 0.001$, $p > 0.10$; AIC $= -10.56$). Models that either deleted genetic influences or the influence of a lifetime health condition on self-reported health produced significantly poorer fits to the data ($p < 0.001$). Figure 1 partitions the variance for self-reported health under the best-fitting univariate model. Figure 1a shows the proportion of the variance of self-reported health attributed to additive genetic effects (A) and unique environmental influences (E) and corresponds to the best-fitting model results of Table 3a. Figure 1b illustrates the proportion of variance in self-reported health as a result of additive genetic influences (A), self-reported health condition (B), and unique environmental influences (E). Figure 1b corresponds to the model-fitting results in Table 3b.

In Figure 1, Model 1a, additive genetic effects account for nearly 40 percent of the variance in self-reported health. The remaining variance is a result of unique environmental influences and error. In Figure 1, Model 1b, it

Figure 1: Variance component estimates percentage (95 percent confidence interval) under best-fitting genetic model for individual differences in self-reported health status. Model 1a does not allow for influence of health condition. Model 1b allows for influence of self-reported health condition. A – additive genetic factors, B – self reported health condition, E – unique environmental factors.



is clear that genetic effects continue to play an important role in self-reported health, accounting for approximately one-third of the variance. Another 15 percent of the variance in self-reported health is accounted for by self-reported health condition. Finally, slightly over half of the variance in self-reported health is attributed to unique environment and error.

DISCUSSION

This report concerns a very simple heritability analysis of one of the most common, reliable, and valid measures of self-reported health in the field. The data were taken in 1987 from the male-male twin pair members of the VET Registry. We found that approximately 40 percent of the variance of the phenotypic response pattern for self-reported health is attributed to genetic effects, with the remainder attributed to unique environment and error. No family, or shared, environment experience effect was found. When controlling for significant self-reported health conditions since discharge from the military, heritability estimates changed, reflecting the importance of condition status. Such findings suggest that the measure of self-reported health is tapping a biologic

dimension, as had been readily reported in the morbidity- and mortality-related literature. The model fitting indicates that a best-fit model can be identified in these data on self-reported health, and it includes a genetic influence for the single-item measure that has not been addressed previously in the literature. The VETS Registry findings differ from the SATSA data reported by the Karolinska Institutet investigators (e.g., no genetic effects for younger age cohorts). These differences may be attributed to different measures, cultural differences in phenotypic responses, or some other unmeasured factors; but it would seem that genetics factors should be considered when using self-reported health as either an independent variable or an outcome variable.

Although not an original intent of the project, we tested whether or not the estimate of the genetic contribution to this global self-reported health

Table 3a: Univariate Model-Fitting Results for Self-Reported Health Status

	Fit of model			
Model	*Chi-sq*	*df*	P *value*	*AIC**
ACE	1.486	0	–	1.486
CE	50.091	1	<0.001	48.091
AE	1.486	1	0.223	−0.541

Table 3b: Univariate Model-Fitting Results for Self-Reported Health Status Allowing for the Influence of a Self-Reported Health Condition

	Fit of model			
Model	*Chi-sq*	*df*	P *value*	*AIC**
ABCE	9.845	9	0.363	−8.155
BCE	49.697	10	<0.001	29.697
ACE	943.928	10	<0.001	923.928
ABE	9.845	10	0.454	−10.155

A = additive genetic effects.

B = self-report of significant health condition since discharge (includes hypertension, respiratory conditions, cancer, heart trouble, stroke, kidney problems, skin conditions, diabetes, gastrointestinal conditions, liver problems, blood disorders, nerve disorders, joint or skeletal disorders, hearing problems, and 'other').

C = shared family environmental influences—influences shared by both members of a twin pair.

E = unique, nonshared environmental influences and error—experiences not shared by both members of the twin pair and error.

* AIC: Akaike's Information Criterion.

could be replicated for specific health conditions. We computed four additional univariate models for high blood pressure, joint problems, stomach disorder, and hearing problems. Because of the lack of adequate power, we were unable to resolve a best-fitting model for the other conditions reported in Table 2, but for these four most prevalent conditions we found that the best-fitting model for self-reported health was similar to Figure 1b (e.g., as a result of genes, the specific health condition, and unique environment with no shared family environmental factor). The variance in self-reported health attributed to genes was consistent across conditions, ranging from 33 percent for joint disorder to 37 percent for hearing condition.

The strengths of this study are tied to its research design that include a large, representative cohort and a widely known, reliable, and valid measure of self-reported health. The VET Registry was identified without knowledge of exposure or disease, and was unbiased in terms of important characteristics (Goldberg, True, Eisen, et al. 1987). The use of twins effectively accounts for many potentially important, but unmeasured, confounding variables, particularly family environmental experiences shared by siblings during childhood and adolescence. Limitations of this study include the male-male twin design that limits our ability to generalize study findings to women and the preponderance of Caucasians, which limits analysis of race effects.

From a behavioral genetics perspective, the results are probably not surprising. From a traditional health services research perspective, the genetic modeling analysis is new and provocative. It is provocative for several reasons. One is because of the magnitude of the variance for self-reported health attributed to genes. The variance estimates for genetic influence is approximately twice the magnitude of the variance attributed to demographic, psychological, and socioeconomic predictor variables of self-reported health in the literature.

The genetic impact on self-reported health would appear to be important for the health services research community. Health services researchers have believed that this indicator of health status was an inexpensive, powerful predictor, but these data suggest at least three avenues for further investigation. First, include more costly, diagnostic evaluative assessments that refine the relationship of biology to self-reported health assessments. Second, explore dimensions of personality with known genetic links (Loehlin 1992) that may further elucidate the genetic influence. The goal of both methods would be to unravel empirically the bio-psycho-social links with health and illness behavior, which is the social context of treatment and outcome. As Coe (1997) suggests, it would seem plausible that because genes and environmental

influences differ for disease groups and help seeking (True, Romeis, Heath, et al. 1997), there are gene and environmental influences that interact with treatments for a desired outcome. Third, we observed that genetic factors accounted for 33 to 40 percent of the variability in self-reported health. This suggests that there may be limitations on the amount of improvement in perceived health status that could be influenced by treatment.

CONCLUSION

This paper is a follow-up investigation on genetic and environmental factors associated with use of health services. We conducted a heritability analysis of self-reported health and found a moderate genetic effect on this strong predictor of use of health services. The heritability findings, coupled with earlier work on the genetic and environmental factors for help seeking, lead to rethinking models of help seeking and health services use. Such a rethinking could follow the suggestion of Mechanic (1994)—that we understand the *etiology* of health services use. For example, the Health Belief model, and its derivatives, would seem to be a good candidate, especially with its interest in preventive behaviors, perceptions of risks and barriers, and cues to action. Another example may imply a fifth iteration of Andersen's (1995) model, where the role of genes and environment would be formally specified and tested. These results suggest a potentially important modification that not only permits modeling genetic and environmental factors for use of health services, but also could easily extend to outcomes.

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